CORRESPONDENCE

Aspartame Disease: A Possible Cause for Concomitant Graves' Disease and Pulmonary Hypertension

To the Editor:

In a recent issue of the *Texas Heart Institute Journal*, Virani and co-authors[1] reported the cases of 2 women, ages 38 and 40, with concomitant Graves' disease and pulmonary hypertension. They also cited 5 reports in the literature since 1999 concerning this combination of conditions.

I have written about aspartame disease for more than 2 decades, because of the profound adverse neurologic, cardiopulmonary, endocrine, and allergic effects of aspartame products.[2,3] These "diet" sodas, along with thousands of other products containing aspartame, are currently being consumed by an estimated 70% of the population. My own database exceeds 1,300 victims of aspartame-related illnesses, with a 3:1 preponderance of women—a difference that is germane to the disorders under consideration.

My 1st report on aspartame-related Graves' disease[4] described 4 weight-conscious women with hyperthyroidism who experienced dramatic remissions within several weeks to 3 months of avoiding aspartame. Four other women who had been treated previously for Graves' disease developed symptoms suggestive of this condition within a few weeks to 6 months after beginning aspartame consumption; symptoms subsided after cessation. These symptoms promptly recurred (generally within 2 days) on multiple rechallenges. The number of patients in this series has doubled since the initial report.

The problem of aspartame-induced pulmonary hypertension was raised by a 27-year-old woman with severe dyspnea and other features attributable to aspartame disease who was found to have primary pulmonary hypertension at autopsy.[5] This association assumed increased relevance because of my database, which revealed that dyspnea was a major symptom in 110 of 1,200 persons (9%) who reacted to aspartame products. In no case could the dyspnea be attributed to a known heart or lung disorder. Most of the patients were weight-conscious women in their 20s to 40s, who experienced marked improvement after avoiding aspartame. Their dyspnea promptly recurred after rechallenge, both upon self-challenge and upon inadvertent exposure.

Aspartame consists of the amino acids phenylalanine (50%), aspartic acid (40%), and a methyl ester (10%) that promptly becomes free methanol after entering the stomach.[3] The breakdown of phenylalanine to highly vasoactive substances—such as dopamine, norepinephrine, and epinephrine—is clearly relevant to pulmonary hypertension, systemic hypertension, and the frequent cardiac arrhythmias experienced by persons with aspartame disease. In my opinion, potent new drugs aimed at reducing pulmonary hypertension should be administered in a patient consuming these products only after a trial of aspartame abstinence.

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References

1. Virani SS, Mendoza CE, Ferreira AC, de Marchena E. Graves' disease and pulmonary hypertension: report of 2 cases. Tex Heart Inst J 2003;30:314-5.
2. Roberts HJ. Aspartame (NutraSweet®): is it safe? Philadelphia: The Charles Press; 1989.
3. Roberts HJ. Aspartame disease: an ignored epidemic. West Palm Beach: Sunshine Sentinel Press; 2001.
4. Roberts HJ. Aspartame and hyperthyroidism: a presidential affliction reconsidered. Townsend Letter for Doctors & Patients 1997;May:86-8.
5. Roberts HJ. Aspartame-induced dyspnea and pulmonary hypertension. Townsend Letter for Doctors & Patients 2003; January:64-5.

The above letter was referred to Dr. Virani and colleagues, who reply in this manner:

We appreciate the comments made by Dr. Roberts, who raised the possibility of aspartame-related Graves' disease and pulmonary hypertension. We look forward to the publication of his observations and database. Our literature search failed to reveal any association between autoimmune thyroid disorders and aspartame consumption. The 2 patients presented in our case reports had features consistent with autoimmune thyroid disorders, as manifested by the presence of pretibial myxedema as well as high titers of thyroid-stimulating antibody. Moreover, these patients both denied any intentional weight loss and were not given any dietary counseling as part of the treatment for Graves' disease. We believe that at the time of treatment and subsequent follow-up, they were still consuming the same kind of diet as they had been at the time of diagnosis. Thus far, neither of our patients has had a recurrence of symptoms on follow-up.